REMARKS/ARGUMENTS

Claims 14-39 are pending. Favorable reconsideration is respectfully requested.

Applicants would like to thank Examiner Collins for the helpful and courteous discussion held with their representative on July 1, 2003. During that discussion, the references cited in the Official Action were discussed with respect to the pending claims. In particular, Applicants' representative pointed out that the *CKI1* gene described by Kakimoto is not used as a selectable marker gene, and that Kakimoto describes plant cells which show physiologically abnormal behavior caused by expression of the plant hormone signal transduction gene (i.e., the *CKI1* gene) were not selected. Adding new claims directed to more specific embodiments were also discussed, as noted on the Examiner's Interview Summary form.

The present invention is directed to a vector for introducing a gene into a plant, which comprises:

a desired gene, wherein the desired gene is not a selectable marker gene,

a plant hormone signal transduction gene and a plant hormone synthesis gene together as selectable marker genes, and

a removable DNA element,

where the selectable marker genes are positioned such that they behave integrally with the removable DNA element, and

where the desired gene is positioned such that it does not behave integrally with the removable DNA element.

See Claim 14.

The present invention also relates to a method of expressing a gene in plants, comprising:

introducing into a plant cell a vector comprising a desired gene and a plant hormone signal transduction gene, wherein the plant hormone signal transduction gene is capable of functioning as a selectable marker gene in the plant,

where

the plant hormone signal transduction gene is expressed in the plant cell, and
the expression of the plant hormone signal transduction gene in the presence of the
plant hormone causes physiologically abnormal behavior in the plant cell, and

selecting plant cells which show the physiologically abnormal behavior caused by the expression of the plant hormone signal transduction gene.

See Claim 21.

The present invention also relates to a method of expressing a gene in plants, comprising:

introducing into a plant cell a vector comprising a desired gene and a *CKI1* gene, wherein the *CKI1* gene is capable of functioning as a selectable marker gene in the plant, wherein

the CKII gene is expressed in the plant cell, and

the expression of the *CKI1* gene in the presence of a plant hormone causes physiologically abnormal behavior in the plant cell, and

selecting plant cells which show the physiologically abnormal behavior caused by the expression of the CKII gene.

See Claim 31.

The rejection of Claim 1 under 35 U.S.C. §112, first paragraph, for alleged lack of written description, is respectfully traversed.

Claim 14 specifies that the "the desired gene is not a selectable marker gene." While there is no direct, literal support in the specification for that limitation, the desired gene is

discussed, *inter alia*, at pages 18-20 of the specification. From that description, one would conclude that the desired gene is not a selectable marker gene, since the vector already contains selectable marker genes (see lines 3 and 4 of Claim 14). In view of the foregoing, the specification does support the recitation in Claim 14 that "the desired gene is not a selectable marker gene." Accordingly, withdrawal of this ground of rejection is respectfully requested.

The rejection of the claims under 35 U.S.C. §112, first paragraph, for alleged lack of written description, as set forth at page 3 of the Official Action dated May 7, 2003, is respectfully traversed.

Page 10 of the present specification provides a detailed description of plant hormone signal transduction genes which can be used as a selectable marker gene. In fact, several specific examples of such genes are provided. Just by providing the names of these genes provides the required structural and functional description of these sequences because, as noted in the specification, these sequences are known in the literature. In addition, the ability of these sequences to function as a selectable marker gene is an inherent property of the sequence itself.

Based on the foregoing, Applicants did have possession of the claimed invention at the time the present application was filed. Accordingly, withdrawal of this ground of rejection is respectfully requested.

The rejection of the claims under 35 U.S.C. §112, first paragraph, for alleged lack of written description, as set forth at page 4 of the Official Action dated May 7, 2003, is respectfully traversed.

The present specification provides a detailed description of how to make and use the claimed vector. As discussed above, the present specification provides a detailed description of plant hormone signal transduction genes at page 10, including several specific examples of

such genes. The working Examples of the application at pages 27-45 provide specific details regarding how to make the claimed vector and use the same. Based on these teachings, one skilled in the art can readily prepare and use other vectors within the scope of the claims. The amount of experimentation would not be undue. Since the amount of experimentation necessary would not be undue, the claims are enabled.

The Examiner has asserted that, since the "degradation" of endogenous chemical substances is well known to be essential to the maintenance of homeostasis in all biological systems, the existence of a "detoxification" mechanism against proteins that mediate plant hormone signal transduction would be essential.

However, the Examiner is confusing the "degradation" with the "detoxification." That is, the "degradation" means that a chemical substance is chemically changed to have a lower molecular weight, whereas the "detoxification" means that a chemical substance is chemically modified to thereby remove its toxicity. Accordingly, the "degradation" does not always involve the "detoxification," and in the same manner, the "detoxification" does not always involve the "degradation." Therefore, the above Examiner's assertion is incorrect on this point.

The plant hormone signal transduction gene used in the present invention expresses in plant cells and produces a protein which mediates the signal transduction of plant hormone. The protein produced mediates the signal transduction either directly or by "degradation." With respect to this point, one of ordinary skill in the art can easily understand that the protein is not subjected to "detoxification" in plant cells. As discussed in the previous response filed on January 7, 2002, the protein functions in the signal transduction pathway of plants, is indispensable for growth and differentiation of all plants, and is naturally exists in various types of plant cells.

Based on the foregoing, the claims satisfy the enablement requirement of 35 U.S.C. §112, first paragraph. Accordingly, withdrawal of this ground of rejection is respectfully requested.

The rejection of the claims under 35 U.S.C. §112, second paragraph, is believed to be obviated by the amendment submitted above and/or is respectfully traversed.

The *CKI1* gene is described in detail in the specification. Accordingly, one reading the claims in light of the specification would readily appreciate the meaning of that term.

The term "isopentenyl transferase" is abbreviated as "*ipt*," as described in the specification.

Again, one reading the claims in light of the specification would readily appreciate the meaning of that term.

Based on the foregoing, the claims are definite within the meaning of 35 U.S.C. §112, second paragraph. Accordingly, withdrawal of this ground of rejection is respectfully requested.

The rejection of the claims under 35 U.S.C. §103(a) European Patent No. 0 716 147 (hereinafter referred to as "EP '147") or U.S. 5,965,791 (hereinafter referred to as "U.S. '791") in view of Kakimoto et al. (Science, volume 274, pp. 982-985, November 8, 1996). Since EP '147 and U.S. '791 appear to be part of the same patent family, i.e, each reference contains the same disclosure, these references will be discussed together with citations to U.S. '791.

The applied rejection is U.S. '791 in view of Kakimoto et al. Therefore, in order for the rejection to be sustainable, the combination of the teachings of U.S. '791 and Kakimoto et al. must suggest the claimed vector. For the reasons set forth below, that combination fails to do so.

U.S. '791 describes a vector containing a desired gene and a morphological abnormality induction (MAI) gene as a selectable marker (see the abstract). As recognized

by the Examiner, this reference fails to teach a vector containing a plant hormone signal transduction gene (see the Official Action dated July 5, 2001, at page 9, numbered paragraph 41).

Kakimoto et al. describe a vector in which *CKII*, a plant hormone signal transduction gene, is the desired gene and an antibiotic resistance gene was used as the selectable marker gene (see page 983). This is evident from Nos. 5 and 6 in the References and Notes at page 985 of the reference. As described therein, a cytokinin-independent mutant which was obtained by introducing the desired *CKII* gene was obtained was selected using the resistance to the antibiotic hygromycin as a selectable marker.

One reading Kakimoto et al. would conclude that the purpose of the vector described therein was to express *CKI1*. Therefore, that gene was the desired gene. Therefore, since CKI1 is not a selectable marker gene, Kakimoto et al. fail to disclose a plant hormone signal transduction gene as a selectable marker gene.

One reading Kakimoto et al. would also conclude that the "additional genes of the Ti plasmid into which the plant hormone signal transduction gene was cloned" were present to assist and direct the expression of *CKI1*., not that they were desired genes to be expressed. As discussed above, an antibiotic resistance gene was used as the selectable marker gene in the construct described by Kakimoto et al. Therefore, the reference fails to describe a vector comprising a plant hormone signal transduction gene as a selectable marker gene.

Thus, Kakimoto et al. fail to describe a vector containing (1) a desired gene which is not a plant hormone signal transduction gene and (2) a plant hormone signal transduction gene as a selectable marker gene.

U.S. '791 and Kakimoto et al., considered in combination, fail to suggest the claimed vector. The vectors described in these references fail to suggest a vector which contains a desired gene which is not a plant hormone signal transduction gene and a plant hormone

signal transduction gene as a selectable marker gene. In Kakimoto et al. the desired gene is a plant hormone signal transduction gene, which is not a selectable marker gene. U.S. '791, as recognized by the Examiner, fails to teach a plant hormone signal transduction gene at all. The Examiner states at page 8 of the Official Action dated March 21, 2002, that the motivation comes from "the success of Kakimoto et al. In using a plant hormone signal transduction gene as a selectable marker gene in plants." However, as discussed above, a plant hormone signal transduction gene was not used as a selectable marker gene in Kakimoto et al. Rather, an antibiotic resistance gene was used as a selectable marker in that reference.

The cited references certainly fail to suggest the methods recited in newly-added Claims 21-39. Those claims specify the use of a plant hormone signal transduction gene as a selectable marker for plant cells which express the gene of interest. Such a feature is not described or suggested by the combination of U.S. '791 and Kakimoto et al.

Based on the foregoing, U.S. '791 and Kakimoto et al., taken in combination, fail to suggest the claimed vector and methods. Accordingly, the combined disclosures of these references fail to establish a prima facie case of obviousness.

Moreover, the experimental data set forth in the present specification is striking evidence of non-obviousness. The data demonstrate the unexpected effect that selection efficiency of gene-introduced tissue can be improved by selecting and using the plant hormone signal transduction gene as the selectable marker gene as compared to the vector described in U.S. '791 (see page 22, the first full paragraph; paragraph bridging pages 32 and 33; page 35, the first full paragraph, *etc.* in the present specification). For example, in Examples 1 and 2, the *CKl1* gene is used as the selectable marker gene so that 100% desired gene (GUS gene)-introduced tissue is selected by using the morphology such as multiple buds as the index (see paragraph bridging pages 32 and 33; page 35, the first full paragraph in

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the present specification). On the other hand, in Comparative Examples 2 and 3 using only

the ipt gene (plant hormone synthesis gene) as the selectable marker gene, the desired gene-

introduced tissues are 14% and 0%, respectively, among the tissues selected using the

morphology as the index (see pages 36 and 37 in the present specification). Therefore, the

selection efficiency is much higher using the claimed vector as compared to the vector

described in U.S. '791.

Based on the foregoing, the claims are not obvious over U.S. '791 in view of

Kakimoto et al. Accordingly, withdrawal of this ground of rejection is respectfully requested.

The obviousness-type double patenting rejection set forth at pages 17-18 of the

Official Action dated May 7, 2003 is respectfully traversed.

The claims of the present application are not obvious over Claims 1, 2, and 4-7 of

U.S. '791 in view of Kakimoto et al. for the same reasons that the pending claims are not

obvious over the complete disclosure of U.S. '791 and Kakimoto et al., as discussed above.

Accordingly, withdrawal of this ground of rejection is respectfully requested.

Applicants submit that the present application is in condition for allowance. Early

notice to this effect is earnestly solicited.

Respectfully submitted,

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